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ON THE

ETIOLOGY OF ANEURISM.

Read at the Liverpool Medical Institution.

BY

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On the Etiology of Aneurism. By JAMES BARR, M.B., L.R.C.S. Edin., Medical Officer to H.M. Prison, Kirkdale; Physician to the Stanley Hospital, Liverpool.

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MR PRESIDENT AND GENTLEMEN,—In this short paper I have no intention of entering into an elaborate disquisition on all the causes which give rise to aneurism, but will confine my attention chiefly to what seem to me to be the principal factors in the production of the disease, and will treat of other agents in a more cursory manner.

I may here premise that I shall use the term aneurism in its widest sense, and, with Walshe, will define it as "a local increase in the calibre of a vessel." From this discussion I wish to exclude traumatic aneurism arising from external wounds, aneurismal varix, varicose aneurism, and cirsoid aneurism, or aneurism by anastomosis.

With this limitation, I think it may be taken for granted that aneurism cannot occur in a previously healthy vessel; there must be degeneration or solution of one or more of its coats before any circumscribed increase in the lumen of the artery can take place. I have myself submitted a healthy aorta to an internal fluid pressure of 10 lbs. to the square inch without injuring any of its coats. The etiology of aneurism, therefore, resolves itself into the pathology of those degenerative changes which give rise to weakening or rupture of one or more of the coats of the vessel. Of all morbid processes associated with aneurism in a causal relation, endarteritis stands first, and shall therefore engage our primary attention; but before dealing with this subject it will be well to take a brief glance at the anatomy of the arteries, in so far as it will help us to a clear conception of the pathology of this affection.

Speaking generally, arteries consist of three coats—(1) an external or fibrous coat, composed of white connective tissue and longitudinally disposed elastic fibres; (2) a middle or muscular coat, consisting of short muscular fibres arranged circularly round the vessel. There is also a certain amount of elastic tissue, together with white areolar fibrous tissue, the quantity of which increases with the size of the artery, while the relative proportion of the muscular fibres decreases; and (3) an internal or elastic coat, which is further divisible into three structures, viz., the endothelium forming the lining membrane, and consisting of irregularly polygonal flattened cells; a sub-epithelial layer consisting of a homogeneous connective tissue and star-shaped corpuscles, and it is in this layer that cell-proliferation most frequently occurs; and lastly, elastic layers, composed of longitudinal elastic networks and the fenestrated membrane of Henle.

The inner coat, which we have just described as consisting of three parts, is quite devoid of blood-vessels, and is consequently nourished by imbibition, not from the blood circulating in the vessel, but from the nutritive fluid exuding from the *vasa vasorum*, which are distributed in the outer and middle coats. These little nutrient arteries divide into small branches on the surface of the artery, which enter the external coat, where they form a network, and a few penetrate into the middle coat, and follow the circular course of its fibres.

On account of there being no vessels in the intima, acute endarteritis is a very rare disease, except when it is associated with and secondary to periarteritis. Even in the aorta, where we might expect inflammation of the lining membrane to follow endocarditis, the disease is rare, or at least it is very rarely recognised; and like endocarditis, when it does occur, it appears in scattered patches, and never as a general inflammation. If with Walshe we look upon those "white opaque cartilaginiform patches studding the inner surface of the vessel, as the chronic representations of the lymph exudations of the acute disease," then I think we must admit that the disease is more common than is generally supposed. My own opinion is that you may have a rheumatic aortitis, but I do not look upon that as simply an endarteritis, but as an inflammation affecting more or less all the coats of the vessels, and perhaps, only lastly, the intima.

The want of direct blood supply to the inner coat, while it renders the vessels less liable to acute inflammation, makes them more subject to degenerative changes of a low chronic inflammatory character.

The intima is nourished by imbibition, which must take place in great part during the period of the vessel's repose, *i.e.*, between the pulsations, when the vessel is lax and not stretched. This period may be broadly stated as occupying an equal time to that of distension, but when you get high arterial pressure there is no proper period of relaxation, but merely periods of greater or less distension. This constant straining from high blood pressure soon leads to mal-nutrition of the inner coat, a chronic irritation or inflammation, which gives rise to hyperplasia of the cell elements in the subepithelial layer of the intima. This cell proliferation first appears as small white or yellowish-white patches, which gradually increase in size, and run together, forming larger patches, and these, from want of proper blood supply, soon undergo retrograde metamorphosis, and become more yellow from fatty degeneration. The corpuscles quickly break up, and then you have got semi-solid masses composed in great part of oil globules and cholesterine crystals, and known as atheroma. These atheromatous patches are situated in the deeper layers of the intima, and not only directly impair the vessel's elasticity, but also from pressure and interference with nutrition lead to fatty degeneration and wasting of the media, and thus impair the contractility and stability of the artery. This atheroma may go on softening and increasing in quantity, and may give rise to solution of the inner coat from pressure leading to molecular changes, and then finally be discharged into the vessel, leaving a wound or ulcer, through which the blood can directly act on the middle coat, separating its fibres, and so lead to aneurism. On the other hand, the atheroma may become hard and firm from the deposition of lime salts, which give rise to the condition known as calcification. These calcified masses, like their progenitors, the atheromatous patches, are first situated beneath the endothelium, but after a time this gives way, and the calcareous plates project into the lumen of the vessel. Calcification also leads to loss of elasticity, localized destruction of the inner coat, with wasting of the media, and consequent lessening of the strength and firmness of the vessel, and so is a frequent source of aneurism.

High arterial tension, of which we have been speaking as the cause of these degenerative changes, is produced in one or both of two ways, viz., by incessant resistance to the outflow of blood through the capillaries, or by greater inflow, owing to increased action of the heart, the former being by far the most important factor, and generally governing the latter.

This high tension and its effects are well exemplified in cases of chronic Bright's disease, and in the labouring population, from the resistance to the capillary circulation, owing to over-work and the imbibition of large quantities of malt liquors, which not only raise the tension by increasing what Johnson calls the stop-cock action of the arterioles, but also by keeping the vessels in a state of constant repletion, thus rendering them more readily affected by any sudden strain or increased pressure.

In those of our race who are subjected to an amount of toil inconsistent with the capabilities and maintenance of the integrity of the human machine, we have not only over-work leading to high arterial tension and its consequent effects,—strain of the arterial coats, loss of elasticity and contractility, with impairment of the strength of the vessel,—but we have also the dire consequences of acute strain giving rise to sudden rupture or stretching of one or more of the diseased coats, thus dilating the artery. From these causes, and others which will be mentioned further on, aneurism is a much more frequent effect of over-work than of Bright's disease.

Another strong proof of the potent influence of high blood pressure producing degenerative changes is afforded by the negative evidence of the rarity of atheroma in the pulmonary system, and the positive evidence that when it does occur there, it arises under the condition of high tension, such as happens in obstructive lung diseases, and in affections of the mitral valve, especially mitral stenosis. In these diseases, as the strain is more or less constant in amount, and affects the whole arterial tree, the atheroma is pretty regular and general, and is most marked where the strain is greatest, viz., in the first portion of the pulmonary artery. Again, I have seen well-marked atheroma in the pulmonary veins in a case of great mitral constriction.

A little evidence which might at first sight seem to tell against the view of the atheromatous origin of aneurism, is afforded by the rarity of that affection in cases of emphysema or mitral obstruction, whereas in tubercular disease of the lungs with cavities, there may be very little atheroma; but this is one of the commonest conditions under which pulmonic aneurism arises. The explanation of this apparent contradiction is simple enough: in obstructive diseases failure of the right ventricle takes place sooner than dilatation or rupture of the coats of the vessel, whereas in the case of aneurism in a tubercular cavity, the vessel was involved in the lung inflammation, and had undergone local changes consequent on the periarteritis. Besides, the walls of the vessel are not supported by lung tissue in the latter case.

Aneurism, although associated with those degenerative changes of which we have been speaking, as a result of high tension, is not a constant or even a common sequence of those changes. You may have most extensive and general atheromatous and calcareous degeneration without any aneurism, or on the other hand, you may have aneurism without much arterial degeneration. In chronic Bright's disease, and in the aged, atheroma is very common and extensive as the result of high tension, while aneurism is very rare; but such evidence as this does not disprove the causal relation in which atheroma stands to aneurism. It only shows that atheroma, although a predisposing cause to the disease, is not of itself usually sufficient to give rise to its spontaneous origin; and the amount of blood pressure capable of producing atheroma is not necessarily competent to give rise to the graver lesion of aneurism. When you have got arterial degeneration, then any straining of the coats of the vessel may lead to an increase in its calibre. Such straining may occur laterally from very high tension, and longitudinally in the aorta and its main branches from the statical blood column under high pressure, stretching the vessels, and in the arteries of the extremities from mechanical strain. From what I have previously stated it will be easily gathered that I believe sudden high tension, especially when associated with over-repletion of the vessels, to be a more potent agent in the immediate production of aneurism than chronic high tension. This is the reason that this affection is so common in our labouring population, and in our soldiers, who during drill are subjected to acute strain with constricted chests. In both cases you have got great bodily exertion leading to high tension and straining, and in the former instance the vessels may be over-repleted from imbibition, and in the latter case you have got in addition the deficient pulmonary area for oxidation, giving rise to overloading of the systemic vessels and increased arterial tension.

Dr Carter tells me that he has known an abdominal aneurism to come on immediately after ligature of the femoral artery.

High arterial tension, which we have been considering, affects all the arteries, we might almost say, in direct proportion to their size, the arch of the aorta suffering most; but there are other forms of strain yet to be considered more fully which lead to degenerative changes, and more or less localize the atheroma. Besides the lateral straining from blood pressure, there may be longitudinal straining of the walls of the vessel, and this may occur independently, or be associated with high arterial tension. Atheroma is more frequently marked along the greater curve of the arch of the aorta than along the lesser, not because the tension is higher in that part, but because there is also superadded the brunt of the blood current.

Again, atheroma is more common at joints than in the continuity of the vessel, because there is the brunt of the blood current during flexions, and the longitudinal straining of the coats during extensions, *e.g.*, popliteal aneurism is very common among the Dublin carmen, owing to the vessel getting contracted or shortened under the influence of cold and wet during a long-continued flexion, and then the coats being strained during extensions of the limbs. In attempting to reduce an old dislocation of the humerus, every surgeon knows the danger of producing traumatic aneurism of the axillary artery.

Again, atheroma is much more common in the neighbourhood of large branches than in other parts of the same vessel, and this, I believe, to be readily explained by the following well-known physical law. When a given quantity of fluid has to pass through a certain length of tube in a given time, the smaller the calibre of the tube the greater the velocity of the current but the less the lateral pressure, and the tube might be reduced so much in calibre that the lateral pressure would be reduced to *nil*, and a hole might be made in the wall of the tube without any fluid issuing therefrom. Large branches are equivalent to an increase in the lumen of the vessel, with consequent increase in the lateral pressure, which we have before shown to be a great factor in the production of atheroma.

In a paper which I published in the *Edinburgh Medical Journal* of December 1876, on the effects of over-work on the heart and aorta, I directed attention to the sequence of the vascular changes arising from over-work, and showed that the right side of the heart was only primarily affected in cases of acute strain or prolonged muscular exertion, while in the majority of cases of over-work with constant high arterial tension, the left side of the heart and systemic arteries were first involved. I pointed out the differences between the effects of acute strain and chronic over-work, and of course an over-worked individual may be at any time subjected to acute strain. I have shown how over-work leads to degenerative changes, and then acute strain may rupture or dilate the already diseased coats of the vessel.

The conclusions to which I came as to the sequence of the chronic morbid changes arising from over-work were as follows:—“(1) Hypertrophy, or hypertrophy with dilatation of the left ventricle, and a slight increase in the growth and capacity of the right ventricle to meet the greater requirements of the system: this condition is associated with high arterial tension, increased aortic recoil with greater supply of blood to the heart; (2) chronic inflammation of, and atheromatous changes in, the aorta and aortic valves, and frequently also in the coronary arteries; (3) loss of elasticity and dilatation of the aorta, consequently the blood is not propelled through the coronary arteries with its former momentum, nutrition fails and dilatation of one or both sides of the heart takes place; (4) incompetence of the aortic valves, with reflux of blood during diastole, which tends to produce still greater dilatation of the left ventricle; (5) this extra strain is met by still further compensatory hypertrophy; (6) failure in the compensation, owing to defective nutrition, with still greater enlargement of the left ventricle and perhaps also of the right; (7) overloading of the pulmonary circuit, with or without incompetence of the mitral valves; (8) additional distension of the right ventricle; and (9) dilatation of the tricuspid orifice."

When aneurism occurs the sequence varies, for that affection takes the place of, or is associated with, the third mentioned condition, and the other events may or may not happen. On the other hand, if early incompetence of the aortic valves take place before there is much arterial degeneration, you don't get aneurism, for low arterial tension prevents undue straining of the arterial coats. Just as I said in my paper that "inspiration has a conservative influence over the aortic valves, which to a certain extent counterbalances the effect of muscular exertion on the aorta and left ventricle, and that I did not know an instance, nor had I read of a case, of sudden rupture of the aortic valves arising under conditions where the chest would be fully expanded," so I now say that, where you have got aortic incompetence before there is much degenerative change in the arterial system, you rarely or never get aneurism. I know that in this view I differ from many writers, and some authors assert that great dilatation of the aorta occurs in cases of aortic regurgitation, from the dilated and hypertrophied left ventricle suddenly throwing a large quantity of blood into the aorta, thus straining and dilating its coats, but this is not in accordance with my experience. I do not say that a certain amount of dilatation may, and does, not take place, but in my opinion, in those cases of greatly or aneurismally dilated aortas with aortic incompetence, the degenerative changes and consequent dilatation take place first and the incompetence afterwards. In short, the authors to whom I allude mistake the effect for the cause.

It has been noticed by Stokes and many others on the subject how frequently you get aneurism without any cardiac hypertrophy. It may then be pertinently asked, If high tension give rise to enlargement of the heart, and also to those morbid changes which lead to aneurism, how is it that hypertrophy of the heart is not always associated with that disease? The answer is, that in those cases where the aneurism is due to primary endarteritis as a result of high tension, and there is no cardiac hypertrophy, once the aneurism has fairly formed, the tension falls. The tension falls from the patient not being able to keep it up with over-work and other causes, and the aneurism when large, acts as a blood reservoir, and never at any time offers any great obstacle to the circulation, so that even if there be enlargement of the heart during the early degenerative changes, the compensatory hypertrophy has time to disappear before you have an opportunity of examining the organs on the *post-mortem* table.

We have seen that atrophy and fatty degeneration of the middle coat accompany or follow atheromatous and calcareous changes in the intima, but how far primary mesarteritis or fatty degeneration of the media, when not associated with endarteritis, contributes to the formation of aneurism, is not well determined. My own experience is that it is rarely limited (and so at anyrate is not a frequent source of any circumscribed increase in the calibre of a vessel), but occurs over extensive arterial tracts, and is generally found in alcoholic drinkers of sedentary habits. It is associated with a flabby condition of body, and fatty degeneration of the muscles, is easily recognised by the defective arterial tonicity, and gives rise to a badly regulated blood 'supply, with consequently imperfectly nourished tissues. It is a disease not very amenable to treatment.

We have already referred to the part played by periarteritis in the production of pulmonary aneurism, and no doubt the arteries in other parts of the body are apt to be similarly affected by inflammatory processes in the surrounding tissues.

Syphilis is frequently looked upon as a common cause of aneurism, though, I think, more from that tendency which there is in the present day to ascribe all the ills to which flesh is heir to syphilis, than from any good and sufficient evidence.

It will be doubtless in the recollection of many here that Dr Davidson brought under our notice, about four years ago, some beautiful specimens of syphilitic disease of the cerebral vessels. Dr Davidson looked upon the changes as partly of the nature of a periarteritis, the adventitia showing the ordinary round cell syphilitic growth, which, he believed, led to narrowing of the lumen of the vessel, and the production of a parietal thrombus which became organised. Heubner, on the other hand, looks upon syphilitic disease of the vessels as a specific endarteritis, the cell-proliferation starting in the endothelium. But whatever view be correct, the facts are that syphilitic neoplasms lead to narrowing of the lumen of the vessel, become organised and converted into cicatricial tissue, and never undergo fatty degeneration. Syphilitic changes are essentially fibrous in character, whereas aneurism is usually associated with fatty and atheromatous degeneration. Moreover, Wilks and Moxon have pointed out the rarity of atheroma, and of course also of aneurism, in prostitutes. Aneurism is about three times more frequent in the army than in the navy, not because our soldiers are more syphilised than our sailors, but because they are subjected to greater strain, and wear tight clothing. I do not say that syphilitic changes may not give rise to malnutrition and be associated with high tension, and thus lead to the production of aneurism, but that it is a common cause, or more than an indirect cause, I will not admit.

With regard to alcohol, it undoubtedly leads to degenerative changes, but I am convinced that of itself it will not give rise to the disease in question. I have before stated that it leads to fatty degeneration and loss of arterial tonicity, with all its evil effects, rather than to any circumscribed affection of a vessel. But when you have got a hard worker, and a hard drinker, you have two important factors leading to atheroma. Of all libations, I consider large quantities of malt liquors, such as beer, the worst, on account of their keeping up high tension, and also leading to a state of chronic repletion of the vessels.

Embolism is generally looked upon as an important cause of aneurism, though there is perhaps a little difference of opinion as to the mode of causation. Some hold that you may have a cerebral aneurism on the distal part of the vessel to the embolic plug, owing to great blood pressure from increased collateral circulation, while others, I think correctly, hold that only inflammatory emboli, which set up inflammation or ulcerative changes in the walls of the vessel at the seat of the plug, and thus directly weaken the artery, are capable of giving rise to the disease. Aneurism thus produced is, we might say, always associated with rheumatic or ulcerative endocarditis, generally occurs in the young, and affects both sexes alike.

Aneurism occurs most frequently between the ages of thirty and forty-five, the period of greatest bodily activity, and is vastly more common in males than in females, especially during this period, as men are subjected to much greater strain than women. At earlier ages the comparative frequency in the two sexes is perhaps more evenly balanced, as the causes are other and more general than strain and other mechanical injuries.

In our soldiers and hard-working labouring population, aneurism is very common, and in them the arteries which are subjected to the greatest strain, and exposed to accidents, viz., the aorta and vessels of the extremities, are chiefly affected. On the other hand, in females aneurism of these vessels is especially rare.